

marked by the appearance of a new pulmonary nodule. Notably, however, the adrenal mass remained stable in size and morphology.

CONCLUSION

The absence of mixed androgen/glucocorticoid hypersecretion, combined with the radiographically static nature of the mass, suggests a lower probability of adrenocortical carcinoma. This case highlights that while size is a major risk factor for adrenocortical carcinoma, it must be interpreted in conjunction with hormonal activity, HU, and growth patterns. Identifying these “mimics” helps avoid over-staging lung cancer and ensures patients receive targeted therapy instead of unnecessary adrenalectomies.

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A HIDDEN DIAGNOSIS: DISSEMINATED HISTOPLASMOSIS MIMICKING TUBERCULOSIS IN THE ELDERLY

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INTRODUCTION

Histoplasmosis is a rare opportunistic, inhalation-acquired systemic mycosis caused by *Histoplasma capsulatum*, endemic to Southeast Asia, including Malaysia. Although classically seen in immunocompromised hosts, it is increasingly reported in immunocompetent individuals. Infection is associated with environmental exposures such as bat or bird droppings and soil disruption. Histoplasmosis is a progressive granulomatous disease that can closely mimic tuberculosis and malignancy, making diagnosis challenging.

CASE

An 87-year-old Malay male with a history of treated pulmonary tuberculosis presented with a 6-month history of intermittent fever, anorexia, weight loss, and hypotension. Initial computed tomography imaging demonstrated bilateral heterogeneous adrenal masses (right: 2.2 × 3.7 × 5.0 cm; left: 2.0 × 3.7 × 5.2 cm) with indeterminate washout characteristics. Biochemical adrenal evaluation, including a short Synacthen test, confirmed primary adrenal insufficiency. Extensive microbiological and malignancy workup, including bronchoscopy, cultures, and tumor markers, was non-diagnostic. In view of clinical deterioration and epidemiological risk, empirical anti-tuberculous therapy was initiated; however, no clinical improvement was observed after 2 months. PET-FDG

revealed intensely hypermetabolic bilateral adrenal masses (SUVmax right 16.7, left 13.2) with no other abnormal foci. Non-invasive fungal investigations were negative. Definitive diagnosis was established via computed tomography-guided adrenal biopsy, which demonstrated necrotizing granulomatous inflammation with intracellular yeasts, subsequently identified as *H. capsulatum*. The patient was treated with oral itraconazole and corticosteroid replacement, resulting in significant clinical improvement and planned interval radiological reassessment.

CONCLUSION

Disseminated histoplasmosis is a diagnostic challenge, particularly in frail elderly patients, where invasive procedures may be delayed. It can mimic tuberculosis and malignancy and may lack an identifiable exposure history. Non-invasive tests may be inconclusive, making tissue biopsy essential. Adrenal involvement may result in primary adrenal insufficiency, further complicating the clinical picture. Early consideration and timely confirmation are crucial for appropriate management.

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OVERWHELMING OPPORTUNISTIC INFECTIONS AS THE INITIAL PRESENTATION OF SEVERE CUSHING SYNDROME

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INTRODUCTION

Severe hypercortisolism is associated with profound impairment of both innate and adaptive immune responses, predisposing affected individuals to opportunistic infections. Excess glucocorticoids alter leukocyte trafficking, suppress pro-inflammatory cytokine production, and impair cellular immunity, increasing susceptibility to bacterial, viral, and fungal pathogens. In some cases, severe infections may precede the diagnosis of Cushing syndrome and represent the initial clinical manifestation. Early recognition is important as untreated hypercortisolism can lead to substantial morbidity and mortality.

CASE

A young adolescent male presented with progressive facial fullness and facial hyperpigmentation for 4 months, followed by 1 month of intermittent fever, cough, lower limb weakness, and hallucinations. On examination, he was tachypneic with cushingoid features including moon facies, pigmented acne over the face and chest, and nail bed hyperpigmentation. He was hypertensive and had severe hypokalemia with lymphopenia.